THE AMERICAN REVIEW OF TUBERCULOSIS

JOURNAL OF THE NATIONAL TUBERCULOSIS ASSOCIATION

ABSTRACTS OF TUBERCULOSIS

EDITOR

ALLEN K. KRAUSE, Baltimore, Maryland

ASSOCIATE EDITOR

MAX PINNER, New York City

EDITORIAL BOARD

JOHN ALEXANDER, Ann Arbor, Mich.
J. BURNS AMBERSON, JR., New York City
E. R. BALDWIN, Saranac Lake, N. Y.
H. J. CORPER, Denver, Col.
F. S. DOLLEY, Los Angeles, Calif.
BRUCE H. DOUGLAS, Detroit, Mich.
L. U. GARDNER, Saranac Lake, N. Y.
ROSS GOLDEN, New York City
ESMOND R. LONG, Philadelphia, Pa.
L. J. MOORMAN, Oklahoma City
D. W. RICHARDS, JR., New York City

VOLUME XXXIX

JANUARY-JUNE, 1939

PUBLISHED MONTHLY

AT MT. ROYAL AND GUILFORD AVENUES, BALTIMORE, MD.
BY THE NATIONAL TUBERCULOSIS ASSOCIATION

THE

AMERICAN REVIEW OF TUBERCULOSIS
ABSTRACTS OF TUBERCULOSIS

VOLUME XXXIX FEBRUARY, 1939 ABST. NO. 1

CONTENTS

PAGES

Treatment
c. *Surgical (concluded)* 1–6
Diseases Other than Tuberculosis
a. *Nontuberculous infections of lungs* 6–12
b. *Spontaneous pneumothorax* 13–16
c. *Tumors of lungs* 17–19
d. *Pneumonoconiosis* 19–20

Tuberculin Hypersensitiveness.—Although mortality rates following thoracoplasty are becoming lower, postthoracoplasty reaction and death still exist and it is felt that to pass off the cause of death as cardiac failure or toxaemia is not telling the whole story. There is a similarity of symptoms in approximately half the postthoracoplasty fatalities where in the fatal cases following the initial reaction, instead of returning to normal, the temperature continues to rise, the pulse becomes rapid, cyanosis deepens, dyspnoea becomes marked and death occurs as a rule from 48 to 96 hours. The clinical picture is not that of cardiac failure and postmortem findings, in general, have been of little help in giving an adequate cause of death. Autotuberculinization has been suggested as a cause of the postthoracoplasty reaction. It was thought that this reaction might represent a union of antigen and antibody and if so, desensitization of the host would be expected and the tuberculin skin test should become negative or be decreased following operation. One-tenth cc. test dose of 1:1000 Old Tuberculin was used intracutaneously, on 46 thoracoplasty cases. The patients were tested from 4 to 7 days before operation, and postoperatively when the temperature had reached normal, usually from 4 to 7 days. The testing was repeated at each successive stage of the operation. The tests were read at 24 and 48 hours, recording the area of induration. The cases could be divided into a group reacting mildly following thoracoplasty, and another group reacting more vigorously. In the former group, there was little change in skin sensitivity, while in the second group there was a marked decrease in skin sensitivity. Tests prior to the second stage showed little change in the former patients, and a return of skin sensitiveness in the latter. There was a general tendency in both groups to lose skin sensitivity. Cases of the first group were represented by 10 productive type lesions, 4 empyemata, 2 postpneumothorax cases, 2 mixed type lesions, and 2 with large apical cavities having little perifocal reaction. Cases belonging to the second group, with the exception of 3, were of the exudative or mixed type. These observations support the view that the postthoracoplasty reaction is basically one representing a rather violent union of antigen and antibody as a result of expressing tuberculin from a tuberculous lung at time of operation. It is associated in some way with the patients' tuberculin hypersensitiveness. The general rule that might be applied would be that a soft lesion associated with a high degree of tuberculin sensitiveness affords the surgeon a poorer operative risk. From observations on a small series of cases, the reaction appears to be a specific one. It is not believed the autotuberculinization is the only cause of postthoracoplasty mortality but that many deaths previously

ascribed to cardiac failure were in reality due to severe autotuberculinization.—*Studies on Tuberculin Hypersensitiveness. 1. The Relation of Hypersensitiveness to Tuberculin to the Post-thoracoplasty Reaction, W. M. Tuttle, E. J. O'Brien & E. A. Graham, Jour. Thoracic Surg., June, 1937, vi, 544.*—(*L. F. B.*)

Death following Phrenicectomy.—Previous medical literature records 44 deaths following phrenicectomy of which 13 were due to spread of the tuberculous process; 7 each to haemoptysis and pneumonia; 6 to cardiorespiratory disease; 5 to surgical haemorrhage; and one each to asphyxia (lung abscess), pulmonary embolism, pyopneumothorax, pneumothorax and mediastinal emphysema, persistent vomiting and an unknown cause. Two more deaths due to cardiorespiratory failure are reported. In one patient the right phrenic nerve was crushed and death occurred 16 hours later. There was a history of rheumatic fever 17 years previously; dyspnoea, oedema of the ankles, loss of weight, and fatigue with a diagnosis of rheumatic heart disease the last 1 to 2 years, and symptoms of pulmonary tuberculosis the last 6 months. Blood-pressure was 110/80; the heart was 64 per cent larger than normal; in the right lung there was a moderately advanced tuberculous process with moth-eaten cavities; and the trachea was in the midline. About 6 hours after operation some cyanosis was noted and the pulse was rapid and weak. Dyspnoea, cyanosis and tachycardia increased in spite of all treatment. Necropsy was not done but the probable cause of complete cardiac failure was change in the intrathoracic pressure with a shift of the mediastinal structures to the right plus the preëxisting mitral lesion. The second patient had 8 inches of the left phrenic nerve evulsed and death occurred 8 days later. She had had marked symptoms of pulmonary tuberculosis for at least 2 years but no cardiac symptoms and she was not dyspnoeic on walking about the ward. Blood-pressure was 112/83 and the heart and mediastinum were displaced to the left. The right lung contained a small infiltration in the upper one-third but on the left there was a huge cavity extending to the third rib anteriorly and some infiltration below the cavity. Autopsy confirmed these findings and revealed some collapse of the lower lobe. The heart was slightly larger than normal, had some left ventricular hypertrophy, and had a patent foramen ovale. Change in position of the heart may have opened the foramen ovale and in that way precipitated heart failure. Since phrenic surgery has been shown to decrease the vital capacity by as much as 500 to 800 cc., or by 36 per cent, a patient having a vital capacity of only 1000 cc. might be reduced to almost the 500 cc. of tidal air. At the time of operation the patient should be observed closely for signs of respiratory or circulatory embarrassment during the induction of the local anaesthesia and the simple test of the patient's ability to hold his breath should also be used. A two-stage phrenic operation is recommended. The nerve is exposed and injected with novocaine, any accessory nerves are cut, and a black silk ligature drawn around the nerve. The wound is then closed with adhesive and the patient observed a few to 24 hours before the operation is completed. Since the mortality of phrenicectomy is 0.5 per cent it is not a minor operation, and any case with evidence of cardiac damage should be carefully observed for some time and a phrenic nerve operation done only after considering the risk and carefully weighing the advantage to be gained.—*Death following Phrenicectomy, D. A. Cooper & W. H. Erb, Amer. Jour. Med. Sc., July, 1937, cxciv, 19.*—(*E. M. J.*)

Thoracoplasty with Pneumothorax and Pleural Effusion.—One hundred cases of caseous pneumonic tuberculosis in which thoracoplasty was performed are reported. All of these cases fulfilled the following criteria: (*1*) distinct cavitation in the upper and outer half of the lung field; (*2*) unilateral caseous pneumonic tuberculosis with or without an exudative productive or productive lesion on the contralateral side and without active tuberculous laryngitis; (*3*) a preoperative existing pneumothorax with or without a serous effusion, or a serous effusion without pneumothorax; in either case the fluid occupied at least one-third of the height of the thorax; (*4*) preoperative positive sputum and postoperative sputum persistently negative for at least four months following the completion of the thoracoplasty; (*5*) survival of the patient for at least four months following the completion of the thoracoplasty. In one hundred and sixteen previously reported cases of thoracoplasty without pneumothorax or pleural effusion only 10.5 per cent remained positive after the completion of the thoracoplasty, whereas in this present series 24 per cent of the cases remained positive. The cases of this present series were complicated by varying degrees of pneumothorax, from a small pleural pocket to a large pneumothorax with serous effusion. Of the former group 12.5 per cent remained

positive after the completion of the thoracoplasty and of the latter 58.4 per cent remained positive. Between these two extremes were cases with varying degrees of pneumothorax which affected the efficacy of the thoracoplasty to a corresponding degree. The existence of air or fluid between the pleural leaves prevents satisfactory coaptation of the pleurae by thoracoplasty since the parietal pleura will not be able to collapse. The regeneration of the ribs will further hold out the parietal pleura and as the air or fluid is subsequently absorbed the lung will expand to meet the parietal pleura. Therefore, in every case the existent air or fluid should be aspirated before thoracoplasty is begun. If this is not possible because of failure of the lung to expand, the air or fluid should be aspirated at operation, following the removal of the ribs and before the muscles have been sutured. In the presence of a bronchopleural fistula continuous aspiration should be resorted to immediately following operation.—*Thoracoplasty for Caseous Pneumonic Tuberculosis in the Presence of Pneumothorax and Pleural Effusion, L. A. Hochberg & P. N. Coryllos, Quart. Bull. Sea View Hosp., October, 1937, iii, 35.—(A. A. E.)*

Collapse Measures.—The classification of disease, amount of collapse therapy used, and the results of bed-rest and collapse therapy in 1,124 patients at the Michigan State Sanatorium have been previously reported. The kind of collapse therapy used in the three degrees of adult-type pulmonary disease was studied in the 886 (595 discharged and 291 resident) patients who received surgical treatment. Of the 39 discharged patients having minimal disease, 92 per cent received only phrenic surgery; none received pneumothorax alone; and 8 per cent received pneumothorax and phrenic surgery. The 215 moderately advanced cases had the following percentages: phrenic surgery alone, 54; pneumothorax alone, 5; pneumothorax and phrenic surgery, 31; total with phrenic surgery, 92; total with pneumothorax, 40 (one patient had bilateral); major thoracic surgery, 11; thoracoplasty, 4; intrapleural pneumonolysis, 7 (6 of 87 patients receiving pneumothorax); extrapleural pneumonolysis, 5; scaleniectomy, 2.3; intercostal neurectomy, 0.5. The 341 far advanced cases of the discharged series had the following corresponding percentages: phrenic surgery alone, 17; pneumothorax alone, 16; pneumothorax and phrenic surgery, 37; total with phrenic surgery, 78; total with pneumothorax, 73 (10 per cent of the 250 pneumothorax cases were bilateral); major thoracic surgery, 30; thoracoplasty, 17; intrapleural pneumonolysis, 12 (31 of 250 pneumothorax cases); extrapleural pneumonolysis, 8; scaleniectomy, 1.1; intercostal neurectomy, 1.4. Attempts to establish pneumothorax were successful in 84 per cent of the moderately advanced cases as compared to 73 per cent in the far advanced cases. The incidence of intrapleural pneumonolysis was 5 per cent higher in the pneumothorax patients having far advanced disease. The resident series (291) indicates that collapse therapy is either immediately or eventually indicated for practically 100 per cent of patients who remain in a sanatorium for at least 6 months. Of the 13 resident patients having minimal disease, 69 per cent received only phrenic surgery; none received pneumothorax alone; 31 received pneumothorax and phrenic surgery combined. The 98 moderately advanced cases had the following percentages: phrenic surgery alone, 31; pneumothorax alone, 12; pneumothorax and phrenic surgery, 27.5; total with phrenic surgery, 82; total with pneumothorax, 59 (15.5 per cent of 59 pneumothorax cases were bilateral); major thoracic surgery, 30; thoracoplasty, 12; intrapleural pneumonolysis, 17 (10 of 59 pneumothorax cases); extrapleural pneumonolysis, 7; scaleniectomy, 4; intercostal neurectomy, 3. The 180 far advanced cases of the resident series had the following corresponding percentages: phrenic surgery alone, 12; pneumothorax alone, 8; pneumothorax and phrenic surgery, 35; total with phrenic surgery, 84; total with pneumothorax, 78 (35 per cent of the 140 pneumothorax cases were bilateral); major thoracic surgery, 44.5; thoracoplasty, 22; intrapleural pneumonolysis, 23 (33 of 140 pneumothorax cases); extrapleural pneumonolysis, 7; scaleniectomy, 4; intercostal neurectomy, 3. Nearly 18 per cent of the 291 surgically treated resident patients received thoracoplasty as compared to 11 per cent of the 595 surgically treated discharged patients. The incidence of the various collapse measures is given in percentages for the 1124 comprising the entire series: unilateral pneumothorax, 51.6 and 40.6, respectively; bilateral pneumothorax, 9.6 and 7.6; unilateral phrenic surgery, 81.5 and 64.2; bilateral phrenic surgery, 2.8 and 2.2 (747 patients had 927 phrenic operations); intrapleural pneumonolysis by closed method, 6.8 and 5.4; intrapleural pneumonolysis by open method, 1.4 and 1.1; intrapleural pneumonolysis by combined open and closed methods, 0.8 and 0.6; unilateral paraffin plombage, 5.3 and 4.2; bilateral paraffin plombage, 0.6 and 0.4; supraperiosteal bag, 0.2 and 0.2; supraperiosteal mus-

cle, 0.2 and 0.2; thoracoplasty, 13.1 and 10.3 (the 116 patients had 361 operations; in one patient it was partial bilateral, and in 7 no other operation was done); scalenieectomy, 2.3 and 1.8 (eight of these patients are listed as having only phrenic surgery or pneumothorax and phrenic surgery combined); multiple intercostal neurectomy 1.7 and 1.3 (the 15 patients had 18 operations, and 10 of these patients are listed as were the scaleniectomies); oleothorax, 1.2 and 1.0. Bilateral collapse therapy was used in 18.1 and 14.2 per cent, respectively, of the two series. This study leads to the conclusion that a series of patients having approximately 60 per cent far advanced, 30 per cent moderately advanced, and 10 per cent minimal disease will require the following collapse measures for the surgical series and the entire series: pneumothorax alone, 10.5 and 8.3, respectively; total with pneumothorax, 60 and 50; phrenic surgery alone, 33 and 25; total with phrenic surgery, 85 and 60; phrenic surgery and pneumothorax combined, 33 and 25; major thoracic surgery 25 and 20; thoracoplasty, 12.5 and 10. Phrenic operations will suffice for 87.5 per cent of the minimal, 50 per cent of the moderately advanced, and 15 per cent of the far advanced cases. The intelligent selection and use of various collapse measures should decrease the necessity for major thoracic surgery.—*Intensive Collapse Therapy in Pulmonary Tuberculosis. II. A Study of the Indications and Use of Various Operative Procedures in a Group of 1124 Patients, G. L. Leslie & R. S. Anderson, Amer. Jour. Med. Sc., July, 1937, cxciv, 1.*—(*E. M. J.*)

Collapse Therapy Results.—To emphasize the benefits of intensive and early collapse therapy in pulmonary tuberculosis, an analysis was made of the 1,666 patients treated in the Michigan State Sanatorium from June 1, 1930 to June 30, 1934. The 1,124 patients having adult-type pulmonary tuberculosis were divided into two main groups. The first consists of 823 who had been discharged and in them the final classification of their disease as well as the prevention and eradication of cavities and of tubercle bacilli in the sputum can be analyzed, while in the second group which consists of 301 resident patients (all under treatment at least 6 months) only the effect on the sputum and the cavities can be tabulated. In the first group, 186 stayed less than 6 months. Many others had been in the sanatorium for months to years before the more general use of the eight surgical measures (1930). Due to a waiting list some patients with sputum conversion and closed cavities were allowed to go home without the time and exercise necessary for a classification of arrested and apparently arrested disease. Collapse therapy was employed in 43 per cent of the minimal; 82 per cent of the moderately advanced; and 73 per cent of the far advanced cases as compared to the resident group in which it was used in 87, 96, and 98 per cent respectively. The resident group had a higher average of major surgery since more of it was done in 1933 and 1934, and also, because thoracoplasty cases are kept strictly in bed for at least 6 months before exercise is started. Bed-rest was emphasized in all cases and, where indicated, collapse therapy was added. The effect of surgical and nonsurgical treatment is compared in each of the two main groups, and also, the final results for the 1,124 cases are given. Of the 595 discharged surgical cases (72 per cent of 823), 7 per cent had minimal, 36 per cent moderately advanced, and 57 per cent far advanced disease. Cavities were present in 77 per cent (457 patients) and tubercle bacilli in the sputum in 76 per cent (450 patients). Treatment resulted in closure of cavities in 71 per cent (of 457); decrease in size of cavities, 13 per cent (of 457); conversion of sputum, 73 per cent (of 450); resulting total with negative sputum, 79 per cent; classified as arrested or apparently arrested, 55.5 per cent, quiescent or improved, 22.5 per cent, and unimproved or dead, 22 per cent. Percentage values for the 228 (28 per cent of 823) discharged nonsurgical patients were as follows: minimal, 22; moderately advanced, 21; far advanced, 57; cavities, 57; positive sputum, 60.5; closure of cavities, 6; decrease in cavities, 12; conversion of sputum, 14.5; total with negative sputum, 49; arrested or apparently arrested, 26; quiescent or improved, 13; unimproved or dead, 61. Sputum remained negative throughout hospitalization in 88 per cent of the minimal and 47.5 per cent of the moderately advanced cases among the 823. The percentage values for the 291 (97 per cent of 301) resident surgical patients are as follows: minimal, 4.5; moderately advanced, 34; far advanced, 62; cavities, 83; positive sputum, 79; closure of cavities, 72; decrease in cavities, 23; conversion of sputum, 70; total with negative sputum, 76. The 10 (3.3 per cent of 301) resident nonsurgical patients are so few, the number of cases gives a clearer picture than percentages: minimal, 2; moderately advanced, 4; far advanced, 4; cavities, 3; positive sputum, 3; closure of cavities, 1; decrease in cavities, 2; sputum conversion, 0; total with negative sputum, 7 (they had always been negative). Corresponding percentage values for the

1,124 patients are as follows: minimal, 9; moderately advanced, 32.5; far advanced, 58; cavities, 74; positive sputum, 73; closure of cavities, 61; decrease in cavities, 16; sputum conversion, 62; total with negative sputum, 72. A separate study of the 144 (17.5 per cent of 823 and 13 per cent of 1,124) patients who died reveals that 90 per cent had a grave prognosis on admission; 56 per cent were under treatment less than a year, 59 per cent received some collapse therapy, 23 per cent major surgery, 13 per cent died within two months of operation, 81.2 per cent died of progressive tuberculosis, and 5 per cent from nontuberculous causes. Collapse therapy was recommended in 81 per cent of the 1,124 patients and was actually used in 78.8 per cent. It was instituted in some form in 72.3 per cent of the discharged patients, and arrest or apparent arrest of the tuberculosis was secured in 47.3 per cent; favorable results in 67.1 per cent; cavity closure in 57 per cent of cavity cases; closure or decrease in size of cavities in 69.6 per cent; sputum conversion in 59 per cent of the positive cases; negative sputum in 70.7 per cent (28.6 had negative sputum throughout their stay). These results constitute an overwhelming argument in favor of a definite policy of early and intensive collapse therapy for approximately three-fourths of the patients with the adult type of tuberculosis in civilian sanatoria in this country.—*Intensive Collapse Therapy in Pulmonary Tuberculosis. 1. A Study of the Extent and Results of Such a Program in a Group of 1124 Patients, G. L. Leslie & R. S. Anderson, Amer. Jour. Med. Sc., February, 1937, cxciii, 149.*—(*E. M. J.*)

Surgical Closure of Cavities.—Attempts at closure of cavities are limited by the type and extent of disease and by the patient's general condition. Its simplicity and adaptability make artificial pneumothorax the method of choice, and truly surgical procedures are generally only to be considered when it has failed. Total thoracoplasty is essentially a procedure for unilateral, chronic and more or less extensive lesions. More localized surgical collapse can be obtained by partial thoracoplasty, extrapleural pneumonolysis and phrenicectomy. Any form of partial thoracoplasty is confronted with the difficulty in obtaining closure of a large rigid walled cavity in the apical region. Here the standard paravertebral resection reduces the transverse or lateral section of the lung, with the effect of compressing the cavity into the form of a slit or tear-drop. To overcome this, modifications have been introduced to free the pleural dome and upper mediastinum, and improve collapse paravertebrally. Apical thoracoplasty necessitates the entire removal of the upper two ribs, with difficult and extensive dissection. Extrafascial apicolysis, introduced by Semb and Holst, aims at dividing all attachments of the pleural dome to mobilize the apex and allow the periosteointercostal muscle bands to drop, being freed from the scalene and serratus magnus muscles. This extrafascial operation is performed outside the plane of the endothoracic fascia, under local anaesthesia. Extrapleural pneumonolysis is more simple, being performed in the plane of the endothoracic fascia, consisting of blunt or manual separation of fused pleural layers from the deep surfaces of the ribs, the later freeing of the mediastinal surface being surprisingly easy, while removal of the upper two ribs is facilitated by exposure of their deep surface. The disadvantage is that as the collection of serum in the dead space under the scapula absorbs the lung expands. This can be overcome by division of the periosteomuscular bundles and sewing them across the apex but the postoperative reaction is likely to be severe with increased liability to infection. Excision of the transverse processes, while advocated to increase the degree of collapse in the paravertebral gutter, interferes considerably with the erector spinae and involves considerable shock in addition to later resulting in severe scoliosis. In any form of apical collapse not more than 3 or 4 ribs should be removed at a sitting, yet sufficient must be taken to avoid production of a sharp shelf between collapsed and uncollapsed zones, on which the inferior angle of the scapula may catch. Rarely is resection of less than five or six ribs adequate. Extrapleural pneumonolysis and plombage with paraffin wax, which is most used, introduces a foreign body that often extrudes itself, hence its indications are limited. Phrenicectomy, although simple and easily performed, is open to much criticism. It may excellently influence middle and lower zone cavities and adherent basal adhesions in artificial pneumothorax. Views are divided on its utility in conjunction with thoracoplasty. At present the temporary paralysis effected by crushing the nerve is most in favor. Partial or selective collapse generally has the merit of leaving as much functioning lung tissue as possible intact, in case later spread elsewhere should occur.—*Surgery in the Closure of Tuberculous Lung Cavities, T. H. Sellors, Brit. Jour. Tuberc., January, 1937, xxxi, 13.*—(*A. P.*)

Partial Resection of Lower Scapula.—A dead space remains following the destruc-

tion of apical pulmonary tissue and obliteration of this space is essential to complete healing. To conserve as much uninvolved lung and vital capacity as possible a partial thoracoplasty is logical although a more extensive operation may be necessary to collapse the disease. Resection of the lower third of the scapula had been done as an alternative procedure to resection of the sixth and seventh ribs. Enough of the scapula is removed to permit it to fall easily into the space provided by resection of the upper 5 ribs. The operation may be completed in one stage but if active paradoxical motion appears after removal of the first three ribs a two-stage operation is advisable. Five representative cases recorded show satisfactory results.—*Partial Resection of the Lower Scapula as an Aid in Compressing Apical Tuberculous Abscesses and in Conserving Vital Capacity, E. Holman, Jour. Thoracic Surg., June, 1937, vi, 496.*—(*L. F. B.*)

Fat Transplant for Closure of Cavities.—The author describes the technique and reports 12 cases in which a free transplant of fat was used to close bronchopulmonary cavities. Complete healing occurred in 11 cases, was partially successful in 2, and a complete failure in 3. Lattice lung from various causes was dealt with. Two cases were tuberculous and the operation was successful in one of them.—*Free Transplantation of Fat for the Closure of Bronchopulmonary Cavities (Lattice Lung), H. Neuhoff, Jour. Thoracic Surg., October, 1937, vii, 23.*—(*L. F. B.*)

Subtotal Scapulectomy.—Partial resection of the scapula was performed on ten patients at Sea View Hospital. The operation was undertaken for the following reasons: (*1*) the persistence of infection between the scapula and the plane of the regenerated ribs after thoracoplasty; (*2*) osteomyelitis of the scapula; (*3*) for the closure of bronchial fistulae or to fill in a large chest defect; (*4*) to eliminate pain and the limitation of motion resultant from impingement of the scapula on the sixth or seventh ribs in partial upper thoracoplasties in which less than seven ribs have been removed. The results were excellent in the closure of bronchial fistulae and in plastic procedures.—*Subtotal Scapulectomy in the Surgery of Pulmonary Tuberculosis, P. N. Coryllos & M. Weinstein, Quart. Bull. Sea View Hosp., January, 1937, ii, 99.*—(*A. A. E.*)

Bronchomoniliasis. — Bronchomoniliasis is no longer considered a rare condition, although the diagnosis must be made with extreme caution since the organism is frequently demonstrated in the secretions of the upper air passages of normal individuals and particularly in the sputum of patients suffering from chronic pulmonary disease, notably, tuberculosis. Reports of the pathological anatomy of this condition have been few and inadequate, since the infection seldom terminates fatally and only rarely have cases come to necropsy. Bronchomoniliasis may be recognized clinically in one of three forms. In the *mild form*, the patient merely complains of a slight cough without temperature elevation or appreciable physical signs of pulmonary disease. The sputum is scanty and mucoid in character, the cough may continue for weeks or months. Frequent recurrence of symptoms is a characteristic clinical feature. In the *intermediate form*, the clinical symptoms and physical signs are more exaggerated. There is persistent low grade fever, the cough is more troublesome, the sputum is mucopurulent and tenacious and may be abundant. In the *severe type*, two clinical subdivisions may be recognized. In the first, the patient suffering from the mild or intermediate form, may suddenly develop an acute pneumonia involving a wide area of the lung, becoming acutely ill, with elevation of temperature. This, lasting for a week or more, may subside completely or may be followed by an empyema or other complication of acute pneumonia. Pyogenic microörganisms usually play an aetiological rôle in these episodes, while at the same time, the fungus finds a fertile soil for further multiplication. In the second type of the severe form, the patient may run a hectic temperature and complain of night sweats, there is gradual emaciation, loss of weight and strength, attacks of dyspnoea and severe paroxysmal cough, worse at night. The sputum is usually copious, mucopurulent, tenacious, glairy, and often haemorrhagic. The course is chronic with periods of exacerbation. In the final stages of the disease death often follows a gradual failure of the right heart. Roentgenograms merely show chronic bronchitis or bronchiectasis, or, in the severe form, wide-spread shadows indicative of an acute diffuse pneumonia, or soft, irregular, mottled or feathery shadows with peribronchial thickening and infiltration and intervening areas of emphysema. Cavities and thickened pleurae may also be noted. The diagnosis cannot be established by the mere demonstration of the yeast-like organism of the genus in the sputum. The sputum must be obtained directly from the lungs, after taking every precaution to prevent possible contamination from other sources. On close inspection, small, whitish granules

may be observed which represent tiny masses of the organisms. These consist of yeast-like bodies, budding forms, and sometimes branching filaments. Since it is generally agreed that *M. albicans* is the only species of the genus pathogenic to man, the identity of the fungus should be established before significance is attached to the finding. Monilia may exist as a saprophyte in the upper air passages. Under favored conditions, the organism may invade the lungs. Monilial lesions of the skin and mucous membranes elsewhere in the body may be a source of infection. Since the monilia is resistant to drying and may live indefinitely in dust and dry environment, inhalation probably plays the most important rôle in infection (such as infection among tea tasters, chinese coolies working in dust of the tea factories, etc.). The pathology of the early pulmonary lesions has not been described, and perhaps cannot be distinguished from those caused by other forms of inflammation. Possibly, the tenacious, semisaprophyte mildly stimulates chronic irritation and a low grade chronic inflammation results. Superimposed recurrent acute infections favor the more rapid growth of the fungus, and small abscesses or pseudotubercles result. These "mycotic tumors" in most cases probably undergo resolution, in others break down, coalesce and progress, or resolve and heal by fibrosis. Only partial healing is the rule, and long continued suppurative and proliferative pneumonitis results, with scattered areas of fibrosis, bronchiectasis and abscess formation with intervening and adjacent zones of atelectasis and emphysema. There are no specific microscopical lesions. Changes observed include nonspecific granulomatous lesions, the infiltrates being usually entirely of plasma cells, less frequently of polymorphonuclears or mixed cellular types, peribronchial and perivascular round cell infiltration, proliferative endarteritis, and proliferative reaction of the alveolar epithelium, abscesses or bronchiectatic cavities, areas of spreading interstitial fibrosis which obliterate alveoli and cause atelectasis and emphysema of adjacent parenchyma. Foreign body giant cells are occasionally observed. The fungus cannot be demonstrated by H and E stain, but is well brought out by Gram-Weigert staining. Yeast-like bodies are scattered through the lesions particularly in the walls of the abscess and perivascular spaces. Mycelia are encountered less frequently. The regional lymph nodes are invariably infested with the fungus. Experimental pulmonary lesions in animals are best reproduced by intrapulmonary inoculation. The direct trauma results in an acute haemorrhagic pneumonia with wide-spread dissemination of the fungus which invades blood vessels and forms thrombi. Upon the subsidence of the acute phase, mycotic tumors develop—white, firm, well circumscribed or coalescent nodules which often break down to form abscesses filled with soft cheesy material. In these experimental lesions, the organisms are often extremely pleomorphic. Small coccal or long bacillary forms, or long, sometimes branching, filamentous threads with various intermediary forms as well as club-shaped mycelia are seen. Whether bronchomoniliasis constitutes a primary disease entity is uncertain. Evidence rather indicates that monilia is probably a secondary invader in a primary lesion (bronchitis, bronchiectasis, pneumonia) already in existence and that it contributes largely to the continued and progressive activity of that lesion. However since (*1*) iodide therapy is almost specific, there resulting symptomatic improvement, or complete cure, (*2*) systemic dissemination occurs, (*3*) no other aetiological agents are found at necropsy, (*4*) the human lesion can be reproduced in experimental animals, the fungus may, in a limited sense, act as an aetiological agent, given a primary lesion in the lung with attendant changes in the tissue which afford a favorable medium for its unrestrained growth.—*Monilia Infection of the Lungs* (*Bronchomoniliasis*), *K. Ikeda, Amer. Jour. Clin. Path., September, 1937, vii, 376.*—(*A. E. M.*)

Pulmonary Mycosis.—Mycotic infections of the lung resemble pulmonary tuberculosis in many respects. They are often diagnosed as such. Occasionally the two conditions are present simultaneously. Pulmonary mycosis can produce in the lung infiltration and excavation, as well as exudative pleurisy. The focus is more often in the base of the lung than is the case with the tuberculous focus. Mycotic infection is rarely localized to the apex. Both the broncho-alveolar syndrome and haemoptysis are common. There are three stages in the evolution of pulmonary mycosis: the bronchopneumonic, the pleurothoracic and the fistulous. Metastasis may occur at any stage. The lesions may be of three types: the nodular or productive, the pneumonic or exudative, and the mixed. The exudative form is rare in man. The lesions are usually unilateral. The first step in the diagnosis of this condition is to rule out tuberculosis. This is not always easy. There is no pathognomonic clinical or roentgenological finding. Moreover Koch's bacillus may in its morphological variations resemble certain of the actinomyces. The two conditions may be present simultaneously. There are cases in which the tubercle bacillus is a secondary

invader, and others in which the fungus seems to have invaded a tuberculous process. The diagnosis may be confirmed by study of serial roentgenograms taken during an iodine "cure." It is a mistake to believe that the prognosis is always more favorable than that of tuberculosis. The present case is that of a forty-two year old male who when first seen complained of a cough which was productive of mucopurulent blood-tinged sputum, a swelling in the left hypochondrium, chills and fever, and progressive emaciation, all of six months' duration. The onset of the illness had been abrupt with pain under the left nipple. The pain radiated into the back. Two days later the cough and blood-tinged sputum appeared. A roentgenogram was taken which showed a fairly homogeneous density at the left hilum from which lines radiated outward but not all of the way to the periphery of the lung. In spite of the absence of tubercle bacilli in the sputum, the patient was sent to a sanatorium where he continued to have a high fever. The lesion had increased in size when the patient was sent home to continue rest treatment. He grew progressively more ill, and his sputum become more and more purulent. The patient lost forty-seven kilograms of weight in six months' time. When he was first seen by the authors there was a subcutaneous abscess in the left hypochondrium. There was dulness over the middle posterior portion of the left lung with fine superficial râles. There was hyperresonance under the left clavicle. There was a tumor the size of an orange beneath the left costal border anteriorly. It was fluctuant, painful to touch, and localized to the subcutaneous tissue. The contents were aspirated and found to be sanguinous and to contain an abundance of single and lumped bacilli. These microörganisms were slightly bent. Their ends were rounded, and from some of them extended filaments a few of which were branched. Anaerobic cultures developed on the eighth to the tenth day. Similar unsealed cultures produced practically none of the parasites. Surface cultures on ordinary gelatin were negative. Filaments were found in the sputum along with pneumococci. No tubercle bacilli were seen. Both bacilli and filaments appeared in blood cultures on the eighth day. Subcutaneous, intravenous and intraperitoneal inoculations into guinea pigs, rabbits, and rats failed to produce any disease or lesions. It was therefore decided that the causative agent in this case was *Cohnistreptothrix Thibiergi*. The authors believe this to be the second case ever reported of infection by this microorganism in man. At the time the diagnosis was made the entire left lung excepting only the extreme apex and base had become involved. There was also a rounded opacity at the right base in contact with the diaphragm. Iodine treatment caused a definite recession of the pulmonary lesions as visualized in the roentgenograms, but the patient's fever continued hectic. Exitus occurred seven months after the appearance of the first symptoms. Autopsy was not performed.—*Mycose pulmonaire par Cohnistreptothrix Thibiergi avec septicémie et métastases, N. N. Stoïchitza & D. Jonnesco, Arch. méd. chir. de l'app. resp., 1937, xii, 56.*—(*C. L. D.*)

Bronchorrhoea.—Bronchorrhoea was described by Williams in 1841 as "an excessive expectoration of a pint or more of clear, slightly viscid, frothy secretion, which appeared to be natural mucus diluted with a considerable addition of the watery part of the blood, with some portion of its saline matter." He also stated that excessive discharge of this type connotes a noninflammatory lesion of the lung. A case is presented where the bronchial discharge was estimated as between 1,200 cc. and 1,800 cc. and where the microscopical sections obtained at autopsy revealed a papillary form of bronchogenic adenocarcinoma.—*Bronchorrhea, T. L. Bliss, Jour. Thoracic Surg., August, 1937, vi, 660.*—(*L. F. B.*)

Cystic Bronchiectases.—For convenience pulmonary cystic formations may be classified in three divisions. The first group comprises solitary or multiple large cysts involving one or more lobes of the lung. The inner surface of the sac is covered by a layer of flat or cuboidal epithelium overlying a layer of laminated connective tissue which contains smooth muscle fibres; there is usually no apparent communication with the bronchial tree. The second group comprises cases in which the affected part of the lung is composed of numerous minute cavitations; on microscopical examination these seem to represent dilated alveoli and bronchioli. To the third group are assigned those cases which seem to occupy an intermediate position between the two preceding groups. In these cases a large portion of the lung is occupied by numerous thin-walled cavities varying in size between that of a pea and a walnut. The sacs freely communicate with the larger bronchi which often show cylindrical or saccular dilatations. The walls of the cysts contain all the elements of the bronchi including muscle, cartilage, and mucous glands. The pathogenesis of cystic disease seems to be that of congenital maldevelopment of the bronchopulmonary structure. Twelve cases which belong to this third group have been studied at the Sea

View Hospital; of these, five have been reported in this paper. All of these patients had marked physical and radiographic changes in the examination of the chest, but none of them had any particular symptoms referable to the respiratory system. On roentgenographic examination the lesion was found to occupy one or more lobes of the lung. The involved portion presents a "honeycombed" or "spongy" appearance which is due to a system of thin-walled cavities with intervening fine septa. There may be fluid levels in the cavities. Bronchograms invariably showed that the contrast material readily entered the cavities; there was also associated cylindrical or saccular bronchiectasis. There were no demonstrable displacements of the mediastinum or alterations in the thoracic contours. This pathological entity can usually be distinguished from other similar conditions and from pulmonary tuberculosis by the disparity between the degree of involvement and the subjective symptoms.—*Cystic Bronchiectases, D. Reisner & I. G. Tchertkoff, Quart. Bull. Sea View Hosp., October, 1937, iii, 1.*—(*A. A. E.*)

Lung Abscess.—Abscess of the lung, formerly considered rare, now occupies an important place in any study of pulmonary pathology. The term abscess of the lung does not include all suppurative processes involving that organ. It is restricted to those collections of pus which are secondary to an acute process, are foul or odorless in character, are inclosed in a cavity, and invade the parenchyma of the lung by means of suppuration and necrosis. This definition excludes suppurative processes which may develop in malignant growths, hydatid cysts, congenital cysts, or in the infarcts occurring secondary to cardiac disease. It also excludes pneumonic hepatization in which the pus is retained within intact alveolar walls. Likewise diffuse peribronchial suppurations are excluded. True gangrene of the lung which is difficult at times to differentiate from gangrenous abscess is essentially a necrotizing process in which the dead tissue tends to be discharged without suppuration. Gangrenous abscess tends rather to sclerosis and the production of a sharply defined border like an abscess. The old classification of Jaccoud according to the method and course of propagation is no longer of any value. Classifications according to size, odor, character of the pus, presence or absence of a false membrane, etc. are all subject to the objection that they are not sufficiently definitive. The presence or absence of necrosis is a very fundamental point and, were it not for the fact that necrosis is characteristically present in abscess due to the Friedländer bacillus, this might be made a basis for classification. At any rate it is of great importance and must be taken into account in any classification. It has seemed most logical both from the clinical and the pathological standpoint to classify lung abscesses according to aetiology. This method will enable us to distinguish at once two large groups. The first includes the relatively odorless suppurative abscesses. Abscess due to the Friedländer bacillus can be included here, for although the odor of the pus is rather nauseating it is not to be compared with that of the material from cases of the second large group, the putrid necrotic abscesses. Varying degrees of necrosis may be present in abscesses belonging primarily in the first group, but the necrosis never reaches the extremes characteristically seen in cases belonging to the second group, nor is the necrosis the final stage and end result of the process. Putrid abscesses whether they are secondary to aspiration or septic emboli, or result from the propagation of an ulcer of the oesophagus, or even when they appear to be primary in the lung are all the result of the simultaneous activity of spirilla and anaerobic bacteria. It is this essential fact which gives unity to the group. The first group can be separated into three divisions according to whether the abscess is produced by amoebae, pyogenic bacteria, or the Friedländer bacillus. The amoebic abscess is characterized by necrosis accompanied by suppuration and is poorly delimited by a sclerotic process. Of the abscesses of the lung caused by pyogenic bacteria the first type includes those which occur in the presence of a septicaemia or pyaemia. These are the abscesses seen in staphylococcus, tetragenes, and enterococcus septicaemias, and in bacterial endocarditis. They are always small and very numerous. The many foci occurring in a single lobe may become confluent and thus produce a very large abscess. Two processes are involved in the development of these abscesses. First a septic embolus produces an area of infarction. There then follows a specific inflammation caused by the organisms which have been carried there. False membranes do not develop. In favorable cases the abscess may heal following the subsidence of the septicaemia. It is generally agreed that abscess is rarely seen complicating a frank lobar pneumonia, but in spite of the fact that the latter condition seems to be less common at the present time than formerly there are numerous reports of cases complicated by lung abscess and in many of these healing occurred spontaneously. These abscesses of lobar pneumonia lie most frequently in the summit of the lobe, but may

be central, peripheral, or near the interlobar fissure. There may be several small abscesses, but usually there is only one. This may vary in size from a few millimetres to several centimetres in diameter. Rarely an entire lobe may become one huge abscess. The abscess cavity is irregular in shape, and its borders are ragged and friable. The pus is creamy, thick and green, and occasionally more or less haemorrhagic. The cavity is traversed by bronchi and blood vessels. Bits of infiltrated parenchyma adhere to the wall or float in the pus. Away from the centre of the abscess the character of the pus changes to that of a purulent detritus. Surrounding this is a firmer sort of material, tissue saturated with pus. Peripheral to this is a region in which infiltration is no greater than that seen in a lung inflamed to the third degree. There are instances in which the abscess is bordered by a yellow-gray false membrane. When evacuation of the pus has been permitted by the establishment of an opening into a bronchus the cavity may undergo retraction and then persist without suppuration or as an ulcer surrounded by a zone of chronic bronchopneumonia. In some cases the abscess may be completely walled off and its contents become caseous. Stokes believes that retraction may then take place gradually until a small fibrous cicatrix is all that remains. The term abscess of bronchopneumonia is restricted to those abscesses which complicate true bronchopneumonias, that is, the pneumonias of infants, the pneumonias associated with grippe, and those seen in the gassed. Bronchopneumonia habitually and naturally tends to abscess formation and this may occur in the course of any bronchopneumonia. In general these are suppurations caused by the streptococcus alone, or accompanied by pneumococci, staphylococci or enterococci. An essential characteristic of these abscesses is their multiple occurrence. Exceptionally one of them is large, but as a rule they are small and numerous, often appearing like miliary tubercles in the region of bronchopneumonia. They are usually yellow and surrounded by a violaceous border. The small bronchi which were the centres of these abscesses have frequently entirely disappeared. Occasionally suppuration will destroy the centre of a patch of bronchopneumonia and a cavity will be left. Barrier has called such a cavity a vacuole. Abscesses caused by the streptococcus are rarely primary. Most frequently they are secondary and are established by either aerogenous or haematogenous spread from a focus. In the first instance they follow diseases of the upper respiratory tract, removal of tonsils, and dental infection. In the second instance they are the result of pyaemic accidents secondary to postabortion infections and suppurative phlebitis. These abscesses vary greatly in size. The cavity is covered by a false membrane which is thin and velvety in consistence. It contains pus and at times blood clots. The inner surface of the cavity wall consists of inflamed and fragmented alveoli. The elastic fibres are frequently still intact. Occasionally there may be a zone of leucocytes in a network of collagen penetrated by new blood vessels. The latter mingle their contents with the pus. The middle zone of the wall is characterized by sclerosis. An abundance of collagenous fibres is found about the walls of the alveoli, while those blood vessels and bronchi which have been spared are surrounded by a ring of fibrous tissue. The peripheral zone consists of alveoli with fibrinous, catarrhal and congestive changes. Rarely there may occur with these abscesses necrosis of the parenchyma similar to that seen in abscess caused by the Friedländer bacillus. The latter organism produces an abscess characterized by the presence of suppuration and necrosis in nearly equal proportions. The odor of these abscesses is foul. Haemorrhage is usually present in the cavity, in the tissues of the cavity wall, and in the surrounding lung. In the middle zone of the abscess wall there is an infiltration with mononuclear cells, a reaction similar to that seen in the putrid abscess. Acute putrid abscesses (necrotizing putrid pneumonias) are of two types. The first and least common type is the necrotizing pneumonia with multiple small foci. Here the lung becomes a sort of sponge of dead tissue and foetid gas. In the second and more common type large cavities develop in areas of bronchopneumonia. They are rarely solitary. Their localization is determined by the situation of the pneumonia. The size of the cavity is quite variable, its shape is irregular and its walls are ragged and festooned and may contain diverticuli. Across the cavity may be bridges of tissue, the remnants of bronchi and parenchyma. In places there may be no true cavity, only an irregularly branching crevice. The wall of the cavity consists of dead tissue into which the cavity has eroded. The tissue is mixed with the pus and fluid contents of the cavity as the latter grows. Occasionally organization takes place and a thin membrane is formed which covers the surface and insinuates itself into the crevices. The cavity is drained by one or more red oedematous bronchi which secrete a viscid mucus. X-ray studies have shown that large cavities may develop in a very few days. All stages of pneumonia are to be seen in the adjacent

lung along with pneumonic areas which are undergoing sclerosis and organization. The latter appear white to the naked eye. In the nonconsolidated lung, congestion, oedema, haemorrhage and emphysema are seen. The pleura is always more or less involved with false membranes, and serous, haemorrhagic or purulent effusions. Subpleural abscesses may rupture into the pleural space. Interestingly enough the interlobar fissures are not involved. The wall of the cavity consists of an inner necrotic zone in which no structure can be discerned and the dead tissue appears to be stratified. Subjacent to this is a zone of necrosis in which all the tissue is dead; no cells, capillaries or nuclei can be seen, but the structural outlines are preserved. Here there is no tendency to stratification. Next is the zone of necrotizing pneumonia. The tissues in this region stain poorly, the alveolar walls are thickened, filled with round cells, plasma and extravasated blood. The capillaries are engorged. Bits of epithelium have fallen into the alveoli and, mixed with macrophages, lie loose in the free space left by the retracted fibrinoid mass. The peripheral zone consists of tissue which stains well, but which presents a picture of congestion and oedema. These zones interpenetrate. Small elementary abscesses are present as areas of peribronchial necrosis, as little nodules of bronchopneumonia with necrotic centres, or as necrotic foci about an arteriole which contains an embolus. All sorts of anaerobic bacteria are present in the contents of the abscess and in the inner zone of the cavity wall. In the latter situation fusiform bacilli and spirochaetes are occasionally seen, but only spirochaetes are present in the zone of active necrosis. Spirochaetes can be found between the collagenous fibrils, in the alveolar walls, in the vessel walls, and in the tissue of the interlobular septa. Upon this fact is based the belief that the spirochaete is an aetiological agent in the production of these abscesses. If the patient survives a sufficiently long time the abscesses evolve further. The necrotizing process may come to a halt and the ragged walls of the cavity undergo organization. Similarly organization and sclerosis may take place in pneumonic areas where no excavation has occurred. Sectioning of such an area of white pneumonia reveals a dry, chalky white, opaque surface. Remnants of collapsed bronchi and vessels may be present, but the tissue is quite bloodless and completely airless. The alveoli are infiltrated with collagenous fibrils, round cells and fibroblasts. The next stage in this process is represented by gelatinous pneumonia. The surface made by cutting is gray, translucent, firm, but elastic, moist and a trifle sticky. The tissue is relatively bloodless and airless. Alveolar structure has practically disappeared with the development of young fibrous tissue. The duration of a pneumonic process is not a factor in determining such an outcome, for an ordinary bronchopneumonia may persist for two months without exhibiting any tendency to organize. On the other hand three weeks' time is sufficient for the development of a typical gelatinous pneumonia when there is an infection of the putrid type present. The determining factor is the infecting agent, its ability to cause necrosis and sclerosis. In some instances in individual lobules or lobes one or the other process may predominate apparently from the very onset of the illness. The end stage, "organized pyosclerosis," presents a varied picture with sclerotic cavities, areas of chronic pneumonia, secondary bronchial dilatation, and secondary purulent or putrid abscesses. The cavity wall may become as tough as cartilage and spoke-like bands of connective tissue radiate from it. Sclerosis may be incomplete and fibrous tissue ramify and be interspersed between foci of purulent and putrid material. Again most of the lung parenchyma may have disappeared leaving only cavities, dilated bronchi, and enough fibrous tissue to support the whole. Diffuse sclerosis may invade all the structures of a lobe so that there are no lobular outlines, nor any islets of parenchyma visible. Only a gray, shiny, dry and rubbery mass remains. This is perforated by burrowing cavities with black and putrid walls and contents. Other cavities with dusky red walls are the remnants of dilated bronchi. The new fibrous tissue tends to contract and occasionally to become exuberant. The former event leads to dilatation of bronchi, retraction of the pleura, and deviation of the mediastinum. In certain instances the fibrous tissue atrophies and leaves only thin strands between abscess cavities. In the walls of large abscesses may be seen all types of epithelium up to true cylindrical. These represent remnants of bronchial epithelium in various stages of regeneration. Nodules of fibrous tissue resembling miniature gummata occur. Giant cells may be present, but their interpretation is uncertain for not infrequently true tuberculous infection complicates these putrid processes. Endarteritis and periarteritis are seen throughout the sclerotic areas. The bronchi may have sclerotic thickened walls infiltrated with round cells. Their lumina may be contracted, and if the cartilage has disappeared the bronchi may be greatly dilated. Tuberculous infection may be present side by side with the putrid process and not penetrate it, or the tuber-

culous granulations may be disseminated throughout the masses of necrosis. The relationship of bronchiectasis and lung abscess is difficult to define. Bronchiectasis may follow such a process as described above. On the other hand an abscess may occur in a previously bronchiectatic area. Likewise an abscess may occur at a distance from a bronchiectasis which has harbored a focus of infection. In some instances both the abscess and the bronchiectasis appear to have occurred simultaneously. In any given case it is very difficult to be certain of the sequence of events which have led to the condition encountered.—*Les abcès du poumon, Henri Durand, Arch. méd. chir. de l'app. resp., 1936–7, xi, 474, xii, 1, 81, 169.—(C. L. D.)*

Pneumococcus Type-VII Pneumonia.—During the years 1928 to 1935 there were seen at the Harlem Hospital 218 patients suffering from pneumonia due to the type-VII pneumococcus. This group comprises 9 per cent of all cases of pneumonia seen at this institution during the above mentioned period of time. Eighteen of the patients were children. Of the adult patients 78.5 per cent were males. The peak incidence among adults occurred during the 1929–1930 and 1930–1931 periods. A cold in the head or chest preceded the onset of pneumonia in only 20 per cent of cases. Seventy-eight per cent of cases began with a chill and 90 per cent began with pain in the chest. Of forty-one cases not treated with serum the temperature of the patient was constantly elevated in 56 per cent. In 24.4 per cent the temperature curve was diphasic, while in 19.5 per cent it was polyphasic. In general these pneumonias were moderately severe. Eighteen and eight tenths per cent of 149 cases receiving no serum therapy died. The mortality figures for males and females did not differ significantly. Excluding six fatal cases with an associated lethal condition, 37.6 per cent of 194 cases were severe. Bacteraemia occurred in 12.1 per cent of 149 cases. In only one instance did bacteraemia develop following serum therapy, and in that case the only positive culture was obtained postmortem. The complications which occurred in the group which did not receive serum therapy included empyema (6 cases), purulent arthritis, meningitis, parotitis, and pericarditis. Albuminuria occurred in 101 patients. This represents 50.5 per cent of the cases in which the urine was examined. Fifty-two patients with type-VII pneumonia received specific serum therapy. The mortality was 6.2 per cent in 48 cases receiving specific therapy. This compares with 17.1 per cent mortality in untreated cases. Of cases in which bacteraemia was present 70.6 per cent of the untreated group died, while of four treated cases one died. Of 129 cases without bacteraemia and not receiving serum 10.1 per cent died. Of 44 cases without bacteraemia but receiving serum therapy 4.5 per cent died. Of fourteen cases receiving serum before the fifth day of the disease none had complications. Five of thirty-six patients (13.9 per cent) treated on or after the fifty day had complications. Of 149 untreated cases 12.1 per cent developed complications. Other organisms cultured from the sputum, blood, or by pulmonary suction include *streptococcus haemolyticus, staphylococcus aureus, bacterium pestis caviae, bacillus suipestifer, bacterium coli*, and pneumococci of types I, II and III. Except for the haemolytic streptococcus which was recovered from four fatal cases none of the above organisms was present in more than one case of the present series.—*Pneumonia Due to Pneumococcus Type VII (Cooper); Specific Serum Treatment, J. G. M. Bullowa & Evelyn Greenbaum, Arch. Int. Med., August, 1937, lx, 179.—(C. L. D.)*

Pneumococcus Pneumonia.—Of 1,850 cases of pneumococcus pneumonia seen during an eight-year period on one service of a New York hospital, 68 were caused by the type-V pneumococcus. This represents 3.5 per cent of cases. In several instances four or five cases were admitted within a few days. Since 1928 there has been an almost progressive increase in the number of cases with infection by this type. Sixteen cases were admitted in the 1935–1936 season. The type-V pneumococcus has a greater tendency to haemolize red blood cells than other types and it is rarely found as a saprophyte in the mouths of normal individuals. In 50 per cent of this series an upper respiratory infection preceded the onset of pneumonia. In 20 cases there was a history of exposure. One case developing in the ward was definitely a contact infection. Seven cases were in females. Except for the occurrence of 24 cases in individuals in the fourth decade of life the age distribution was fairly even. The onset of the pneumonia was abrupt with a shaking chill, thoracic pain, and cough in 46 cases. Rusty or frankly haemorrhagic sputum was present at the onset in 38 cases. In 22 cases the onset was atypical, and the fever irregular. These cases occurred during the course of upper respiratory infections. In general the fever was of the plateau type. Thirteen of the 18 cases recovering without serum therapy terminated by crisis. Fifteen of 29 cases recovering and receiving serum terminated in the same manner. The duration of illness tended to be long. In only 7

of 18 cases recovering without serum therapy did the fever persist less than one week. In three cases it was present for more than ten days. In 20 cases headache, usually severe, was an early symptom. In one case it was so severe that meningitis was suspected. Vomiting occurred in 19 cases in three of which it was accompanied by diarrhoea. Jaundice was present in 16 cases. This is a very high incidence. Bacteraemia was present on admission in 18 cases and developed later in two more cases. This gives an incidence of 29 per cent which is higher than for any other type of infection. In practically all cases a slight cyanosis was present some time during the illness. It was marked in 5 cases and moderately severe in 11. Estimation of the severity of the illness was based on the presence of bacteraemia and anoxaemia, on the age of the patient, and on the patient's general health. Considering these factors 12 cases were extremely severe, 38 were moderately severe and 18 mild. Serum therapy was used in 39 cases. In only 12 of these was serum given within the first three days of the pneumonia. In 9 cases it was given later than one week after the onset. Two of twelve patients receiving serum within the first three days of illness died. Three of 25 patients receiving serum within the first five days died; and 7 of 13 patients receiving serum after the fifth day died. Empyema complicated 7 cases. In only 2 of these had serum been given. Endocarditis occurred in 3 cases, suppurative bronchitis in 3, and meningitis in 2. Otitis media, pleurisy with effusion, pulmonary abscess, purulent pericarditis, embolism of the subclavian artery, and glomerulonephritis were each present once as complications in this series of cases. The concept that type-V pneumonias are always mild is certainly belied by this series.—*Pneumonia Due to Type V Pneumococcus, M. B. Rosenblüth & M. Block, Arch. Int. Med., October, 1937, lx, 567.*—(*C. L. D.*)

Fatal Spontaneous Pneumothorax Due to Ruptured Bulla of Lung.—Spontaneous pneumothorax occurring suddenly in apparently healthy persons is occasionally reported and, being rarely fatal, opportunities for observing the nature of the underlying lesions are few. In cases coming to necropsy it has sometimes been traced to ruptured subpleural bullae. In this case the clinical symptoms were unusual and chiefly referred to the abdomen. *Case Report:* A coal miner of 29, while shovelling underground, was suddenly seized with upper abdominal pain and admitted to the hospital in a state of shock with shallow respiration and right upper quadrant tenderness and rigidity. As a perforated peptic ulcer was suspected a laparotomy was performed, with practically negative findings. Next day there was right chest pain and very rapid shallow respiration. Physical signs indicated pneumothorax and 7 litres of air were withdrawn over an hour's time with temporary improvement but later death. Necropsy showed an extensive right subcutaneous emphysema of the chest and complete collapse of the right lung. At its apex was a typical healed tuberculous lesion, with a fibrous pleural scar and puckering of adjacent lung tissue whence a cord-like adhesion an inch long extended to the dome of the parietal pleura. Near its pulmonary point of attachment was a collapsed bulla of somewhat smaller diameter through which water escaped when the submerged lung was perfused. The opposite lung was oedematous. As there were bubbles of air beneath the liver capsule their entrance was doubtless by way of the falciform ligament.—*Fatal Pneumothorax Due to Rupture of a Solitary Bulla of the Lung, J. Gough, Lancet, August 7, 1937, ccxxxiii, 314.*—(*A. P.*)

Atypical Spontaneous Pneumothorax.—Classically spontaneous pneumothorax is distinguished by a definite symptomatology. This is true whether the onset is abrupt or insidious. In the former instance pain, dry cough, and dyspnoea are usually present. Cases with atypical clinical manifestations are not infrequent. Syncope may accompany the onset as in the case of Chabaud. That patient had done his morning setting-up exercises and was shaving when he felt a not particularly painful sensation as if something were passing through his chest. At noon after a two hour walk he returned home and lost consciousness for several minutes. There had been a right sided pneumothorax. Another case has also been reported in which the onset was accompanied by convulsions. Cases simulating angina pectoris and coronary occlusion have also been met with The authors report the case of a thirty-two year old clerk who one morning while climbing a flight of stairs in his home was suddenly seized with a violent pain in the region of the precordium. The pain radiated along the left arm and into the last two digits of the left hand. Cold perspiration, anguished facial expression, sense of constriction in the chest, and pallor were all prominent. The physician who saw him thought first of a coronary occlusion, but physical examination of the chest revealed the true nature of the trouble. Roentgenograms confirmed the presence of a pneumothorax. In another of the authors' cases, localized precordial pain

was present and accompanied by very deep respirations. The patient lay on his left side with the legs flexed. Such cases it would seem best to classify as reflex angina. This would seem to be consistent with the recent work of Katz. In the authors' experience, cases with symptoms simulating brachial neuralgia have been associated with a frank thoracic symptomatology as well. The least common form seems to be that simulating pericarditis. Such was the case of Deomeules. A man twenty-two years of age and suffering from pulmonary tuberculosis was suddenly seized with a violent pain in the anterior inferior part of the left chest. Weakness, pallor, and restlessness were accompanied by pulsation and hyperaesthesia of the precordium. This proved to be a case of pneumothorax localized to the lower third of the left chest. Forms simulating the pain of acute abdominal conditions may occur. The authors report a case with pain of rapidly increasing intensity localized in the epigastrium and the right iliac fossa and radiating upward. In another, the pain was confined to the lower part of the sternum. In a third case the pain was thoracic, but there was frank resistance to palpation in the region of the right hypochondrium. A fourth case was unusual in that the pain began in the epigastrium, was of a stabbing nature, and radiated to the back. It was accompanied by a sense of suffocation and by collapse. In this case, examination of the abdominal wall revealed an absence of the respiratory excursion, and retraction of the abdominal wall. Both palpation and percussion produced pain. Several cases of this type have been reported in the literature. The pain may reproduce that of acute inflammation of various abdominal viscera. It may be accompanied by what appears to be a peritoneal reaction, with nausea, vomiting, rigidity, or defense spasm of the abdominal muscles, changes in the facies, pulse and temperature. Careful physical examination of the chest is the most important means of arriving at the true diagnosis. Forms simulating chronic abdominal disease also occur. Schreiber has reported a case of a patient who consulted him for digestive symptoms. Discovery of a left pneumothorax led to appropriate treatment which cured both the pneumothorax and the indigestion. In view of the studies of Pottenger the origin of the pain in the anginal and abdominal forms is best explained as due to sympathetic viscero-sensory reflexes beginning in the pleura. Muscle spasm would accordingly arise from sympathetic viscero-motor reflexes. While cough and vomiting would be produced by parasympathetic reflexes.—*Formes douloureuses insolites du pneumothorax et de l'hémopneumothorax spontané, M. B. Castex & E. S. Mazzei, Arch. méd. chir. de l'app. resp., 1937, xxi, 111.*—(*C. L. D.*)

Spontaneous Benign Pneumothorax.—The great frequency with which pulmonary tuberculosis is responsible for the occurrence of spontaneous pneumothorax led to the idea that in the absence of other frank pulmonary lesions such as neoplasm, lung abscess, etc. all cases were to be ascribed to tuberculosis. This soon resulted in the necessity for recognizing "special" forms of pulmonary tuberculosis; forms without fever, tuberculosis of the pleura without exudation, subpleural tuberculosis without any roentgenological evidence, and with a negative reaction to tuberculin, and finally cases in which the pathological anatomy was found to be completely lacking in specific lesions. Further the very prompt and complete recovery in such cases was inconsistent with a tuberculous aetiology. As final evidence of the benign nature of this type of accident came the reports of cases with four and five recurrences but never developing roentgenographic evidence of pleural adhesions nor of any other sort of inflammatory process. The present paper summarizes twelve cases of benign spontaneous pneumothorax all of which demonstrate the truly benign nature of the condition. This study indicates that the condition occurs chiefly in males (there were no females in this series) between the ages of twenty and thirty. Nine of the cases were in the third decade of life and the other three were between thirty and thirty-five years of age. There is no reason to suspect a familial tendency to spontaneous pneumothorax. Onset may be acute or insidious, and may occur during violent physical exertion or during rest. Pain and dyspnoea are the cardinal symptoms. Cyanosis, nausea and vomiting, cold perspiration, and palpitation may be classed as the secondary symptoms. There is no febrile reaction. The pain is variable in its intensity; it may be localized or radiate over the thorax or abdomen. In general it may be stated that physical examination will reveal an increase in size of or an immobility of one side of the thorax, decreased or absent tactile fremitus on the involved side, and tympany of a higher or lower pitch depending upon the tension of the trapped air. Breath-sounds are diminished or absent, or they may be amphoric in character. The heart is more or less displaced, but soon returns to its original position. Roentgenograms confirm the diagnosis. The pneumothorax is usually total, because the patients are otherwise in good health and do not have

pleural adhesions. Subpleural bullae may be seen as annular shadows with narrow margins. Their demonstration is largely dependent upon their location. They can usually be seen if situated in the apex, or on the anterior or posterior surface of the lung, or marginally. The number present and the size of the bullae are quite variable. They were demonstrated in roentgenograms from three of the present series of cases. In spontaneous pneumothorax the intrapleural pressure tends to be negative or to fluctuate about zero. Exceptionally it is 0 to +10. The physical signs usually return to normal by the end of fifteen or twenty days, and complete recovery rarely takes over twenty-eight days. The complications occasionally met with include intrapleural haemorrhage, and simultaneous pneumothorax of the contralateral side. Pneumothorax may be recurrent. It may be bilateral either simultaneously or successively. It is rarely chronic or permanent. The treatment is essentially bed-rest, sedation, and if necessary aspiration and administration of oxygen. Exact knowledge as to the pathogenesis of this condition has been hampered by its very benign nature. As to the subpleural bullae they may occur anywhere over the surface of the lung. They vary in size from that of a millet seed to that of a hen's egg, and may be solitary or multiple. In general their bases will be found to have a cicatrix of the type commonly believed to be secondary to an inflammatory process. Their aetiology, however, is still a matter of debate. They may be secondary to a congenital malformation or to an inflammation. No one has found any definite evidence pointing to tuberculosis as a factor in the production of this condition. The bullae have been observed by means of the thoracoscope in cases of benign spontaneous pneumothorax both by the authors, and by Wiele, and Cova.—*Le Pneumothorax spontané bénin par rupture des bulles sous-pleurales, M. R. Castex & E. S. Mazzei, Arch. med. chir. de l'app. resp., 1937, xii, 23.*—(*C. L. D.*)

Recurrent Benign Spontaneous Pneumothorax.—The authors first review all cases of recurrent benign spontaneous pneumothorax previously reported. These cases had varying numbers of recurrences ranging from one to over fifteen. Three new cases are then reported. The first is that of a young man who from his twenty-first year suffered from a series of spontaneous pneumothoraces. They occurred always on the left side, and were characterized by pain and dyspnoea, and absence of fever. There was never any evidence of tuberculous infection in this case, and roentgenograms of the chest taken after the ninth episode failed to demonstrate any pleural adhesions. The second case is that of a chauffeur, twenty-five years of age, who immediately after cranking his car was seized with a pain in his right chest. He described it as feeling as though a dagger had been thrust into him. He immediately became dyspnoeic. Roentgenogram of the chest taken some time later showed a complete pneumothorax on the right side. There was no febrile reaction and the sedimentation rate was not elevated. Recovery was uneventful. Four months later while again in full health he suffered a similar episode. The right lung was again involved. The course was as benign as previously, and there was no evidence that might point to an infectious aetiology. The third case was that of a man who suffered his first attack at the age of thirty-four. He was awakened one night from sleep by a dry cough and a sense of suffocation. Roentgenograms showed a left pneumothorax. The patient had no fever. Six months later a similar episode occurred in the early morning hours. Two years later he was again wakened with a sense of suffocation and of impending death. This was soon followed by nausea and vomiting, but by evening the symptoms had subsided, only to reappear at 10 p.m. while the patient was raising himself in bed. Recovery was as usual uneventful. One morning fourteen months later he was awakened with a pain in his back, chiefly on the left side, and accompanied by cough. As there was no dyspnoea present this time the patient went to his work, but suddenly his symptoms grew more severe requiring his return home. The last of this patient's episodes occurred fifteen days after recovery from the one just mentioned. All of these involved the left side, and were unaccompanied by fever. There is no reason to think that these recurrent cases differ greatly from the ordinary case of benign spontaneous pneumothorax. They occurred in young men who had been in good health and who maintained good health after the acute episodes had passed. There was no question of tuberculosis or of chronic emphysema. There was no pleural exudate nor any fever. The recurrences were all on the same side. In considering the aetiology of this condition attention is called to Gunther's report of the finding of many cyst-like subpleural dilatations at autopsy in such a case. Castiglione and Cova after observing through the thoracoscope subpleural dilatations in a case of spontaneous pneumothorax were later able to verify their observations at the autopsy table where these were seen to be subpleural sacculations filled with air. All roentgeno-

grams from one of the cases here reported showed consistently annular shadows which could represent bullae. It was shown experimentally by the inflation of cadaver lungs that it was at the sites of such bullae or where for other reasons there had been some elevation of the pleura that perforation took place. Further, Hohenner has shown that near the hilum of the lung there is a weak point where with increased intrapulmonary pressure, interstitial emphysema will occur which may be followed by spontaneous pneumothorax. He was also able to show that cicatricial apical bullae, alveolar emphysema and congenital malformations of the lung can be the cause of weak spots in the lung tissue, but that changes localized to the pleura and unassociated with parenchymal change will not cause pneumothorax. Roentgenographic studies show that the opposite lung behaves as with therapeutic pneumothorax. There is exaggeration of the bronchovascular markings. This disappears as the air is resorbed from the pleural cavity of the involved side. The treatment of these recurrent cases is similar to that of simple spontaneous benign pneumothorax. Attempts have been made to prevent recurrences by stimulating the development of pleural adhesions. Instillation intrapleurally of dilute silver nitrate solutions and of 50 per cent dextrose has been tried.—*Pneumothorax spontané bénin récidivant, M. R. Castex & E. S. Mazzei, Arch. méd. chir. de l'app. resp., 1937, xii, 39.*—(*C. L. D.*)

Spontaneous Haemopneumothorax.—This condition has been reported since the time of Littré, and it is probably more common than the 49 cases reported since 1900 would indicate. Of the 44 cases in which the age was stated, 26 were between 20 and 30, and 16 between 31 and 40 years of age. Only one patient (one of this series of three) was a woman. Of the 49 cases, 8 died, and 2 of these had tuberculosis. Only 6 others of the series had tuberculosis. The following 3 patients were all 30 years of age and able to work. They had no roentgenological or bacillary (sputum and pleural fluid) evidence of tuberculosis during their hospitalization, or on repeated X-ray examinations 24, 11 and 6 months, respectively, following haemopneumothorax. Case 1 was a colored man who had a cold about a week before he choked on food causing him to cough several times. Thoracic pain (left) gradually became severe within an hour. On hospitalization 24 hours later he had severe pain, some dyspnoea but no cyanosis. Respiratory rate was 32 per minute, pulse rate 132, blood-pressure 114/80. Physical examination revealed complete pneumothorax with a small amount of effusion. Twelve hours later there was extreme dyspnoea, blood-pressure 94/70, and physical and roentgenological evidence of massive effusion. Intrapleural pressure was plus 4, and 1,600 cc. of air followed by 80 cc. of bloody fluid were allowed to escape. Dramatic clinical improvement occurred and blood-pressure rose to 118/82. The capillary blood had 4,300,000 erythrocytes and 90 per cent haemoglobin while the pleural fluid had 80 per cent haemoglobin. During the next month aspirations (2,800 cc. fluid containing about 1,250 cc. of blood) were necessary to prevent dyspnoea and the capillary haemoglobin fell to 63 per cent. Complete re-expansion occurred in 7 weeks. Case 2 was a white woman who developed malaise, chilliness, fever and slight cough for which she stayed in bed one day. Four days later, with no associated straining or coughing, she had sudden, sharp thoracic (left) pain which persisted. On hospitalization, five days later, partial pneumothorax and a small amount of effusion were found. She was not dyspnoeic but 150 cc. of air followed by 120 cc. of bloody fluid were aspirated. In three weeks complete reëxpansion had occurred. Case 3 was a white man who, several years ago, had had sharp thoracic (left) pain lasting a few hours. About 30 hours before admission he had, while shaving, sudden, sharp, severe pain in the base of the left lung. In a half hour he felt better and went on to work. The pain again began to increase in severity and five hours later was suddenly more severe, causing him to collapse but not to lose consciousness. Severe dyspnoea persisted but the pain was somewhat relieved by strapping. On admission hydropneumothorax and possible apical intrapleural adhesions were found. Blood-pressure was 108/65; pulse, 108; and respiratory rate, 30 per minute. Three days later, 300 cc. of bloody pleural fluid (4,500,000 erythrocytes) were aspirated. In about two weeks the lung was practically re-expanded. The initial attack of pain probably represents the onset of pneumothorax which is most likely due to the rupture of a superficial air vesicle while the second pain is probably due to rupture of an adhesion with accompanying haemorrhage. Symptoms depend on the size of the vessel torn and whether or not there is a valve-like action in the ruptured vesicle. Aspiration of air and blood should be done only for dyspnoea during the first 48 hours. When the bleeding has stopped, the fluid should be replaced by air. There is no adequate explanation for such a marked predominance of males among those affected.—*Spontaneous Hemopneumothorax. Report of Three Cases*

with a Review of the Literature, H. U. Hopkins, Amer. Jour. Med. Sc., June, 1937, cxciii, 763.—(E. M. J.)

Benign Tumors of Bronchi.—This paper deals with solid epithelial tumors of the bronchi and does not include papillomata, fibromata, lipomata and the like. The present study includes twenty-three cases from the literature and nine cases which the author had available for detailed presentation. Most of the cases occurred between the ages of thirty and forty, nine cases, however, were from the third decade of life. Because symptoms may not appear early in the development of these slow growing tumors, it is quite likely that the tumors originate even earlier in life. Both sexes are affected in approximately equal proportions. The tumors are either pedunculated or are situated within the wall of the bronchus and project into the lumen in varying degrees according to the layer of the bronchial wall in which they have originated. Their diameter varies from a few millimetres to a few centimetres. Sectioning reveals a smooth pulpy, gray-red surface which is rather soft. The tumors are usually round and unless pedunculated are surrounded by a capsule. In all cases they have a smooth outer surface. Mucous membrane more or less completely covers the exposed surface of the tumor or may be present only around the base of the pedicle. Not all of the layers of the mucous membrane can always be identified, and often only one is present. This may consist of mere connective tissue ramified by elastic fibres and surrounding the tumor like a capsule. Occasionally isolated bits of tumor are found in this capsule, but never in abundance, nor do they penetrate into and through it extensively. The absence of various layers of the mucous membrane is readily explained by the fact that this tissue is easily exfoliated under ordinary conditions, and when it is pushed up by the tumor it is more susceptible to trauma. In fact even the tumor tissue itself may be eroded. The submucosa is penetrated by tumor tissue in varying degrees. It may be more or less completely atrophied with only here and there a remnant persisting. The base of the polypoid forms may penetrate to the cartilage of the bronchus. This layer is regularly penetrated by tumors situated within the bronchial wall. In such instances the cartilage shows signs of disintegration, and only the bony constituents may remain. The tumor may even spread out to involve the connective tissue about the bronchi, but it never involves the pulmonary parenchyma or the lymph nodes. It is surrounded by a connective tissue capsule which it never penetrates. Tumor cells are not seen in the lymphatics. In one instance tumor tissue was found in the lumen of a small vein. Pedunculated tumors occur chiefly in the larger bronchi. Tumors of the main bronchi cause diminution of the lumen and produce respiratory disturbances. Secretion is retained behind the tumor, and unilateral bronchiectasis results with inflammation and a chronic pneumonia. This may be the first sign of illness. In some cases the chronic suppuration has led to amyloidosis. If only a small bronchus is involved atelectatic induration may result. Tumors situated deeply in the bronchial wall may appear first as incidental findings at autopsy. But even these deep-seated ones may haemorrhage from time to time if their surface is eroded. Symptoms may be present for years before a correct diagnosis is made. One should always bear this possibility in mind when confronted with a case of frank unilateral bronchiectasis. Bronchography and bronchoscopy should be resorted to and the obstruction revealed. The polypoid forms can be removed and the patient cured. The first two of the author's cases and that of Boemke are clearly cylindromata. There was a deposition of hyaline and mucous material not only between the tumor epithelium, but also between the latter and the supporting structure. Cylindromata occur elsewhere in association with mucous, serous or mixed glands. It is therefore fair to assume that these tumors arose from the bronchial glands. Boemke believed the tumor in his case was malignant because it had penetrated the entire bronchial wall. In both of the present author's cases this had also occurred. They were all three probably cases of benign tumor. The other seven cases resembled those described by Geipel. Two cell types could be distinguished. Many groups of cells consisted solely of palisade cells arranged in cords and lying perpendicularly to the course of the cord. These cells have oval nuclei whose long axes lie perpendicular to the direction of the cord. The cell bases are broad and lie against the stroma while the clear tapered ends are interlocked between the bases of the opposite row of cells, but frequently reaching the stroma on the other side of the cord. The other cell type commonly present is the smaller more polygonal cell. These cells have rounded nuclei rich in chromatin. They lie irregularly against one another, and may also form cords. The outlines of these cells are not so clearly defined. Both cell types, as a rule, partake of the structure of the tumor. Occasionally the nuclei of the palisade cells are more distant from the cell base and in this manner there is produced a broad strip

of protoplasm along the edges of the cords. In places the cells are arranged in spoke-like fashion about a minute space which contains a drop of mucus. Elsewhere the arrangement resembles that which is seen in carcinoid of the appendix and which is spoken of as "évolution cylindromateuse." Finally there may be groups of onkocytes. All of these cell types and arrangements may be found in some tumors, rarely does one greatly predominate. The framework is in general poorly developed, and consists of coarse septa rich in collagenous fibres and containing nutrient vessels. It terminates as a capillary poorly surrounded by connective tissue. What elastic tissue is present represents remnants of that of the bronchial wall. The stroma is never infiltrated. Although these tumors penetrate the bronchial wall and destroy its tissue, they never produce the amount of necrosis that a cancer does. The cells are grouped in a more or less orderly fashion, but at times, especially with the small polygonal cells, their variation in size is suggestive of cancer. Mitosis is rarely seen. Many cases reported in the literature as polypoid bronchial carcinoma have probably been benign. Certainly this is the case with one in which the patient died eleven years after coughing up the bronchial tumor which was called malignant after histological study. At autopsy no evidence of metastasis was found. There are no proved cases of such tumors giving rise to carcinoma. Except for the presence in these tumors of palisade cells they resemble in arrangement of cells and in the cell types the rare solid epitheliomata or adenomata of the intestinal glands. It is possible that the palisade cells originate not from the bronchial glands but from the cylindrical epithelium lining the wall of the bronchi, which has become incorporated in the tumor. The result is strikingly like that seen in carcinoid of the appendix. In none of the bronchial tumors, however, were there spindle cells, nor do carcinoid tumors contain onkocytes. The lack of stroma reaction, the presence of little gland-like empty spaces, the grouping of palisade cells about capillaries, the impregnation of the connective tissue with a mucous material, and the tendency of the tumor to penetrate the layers of tissue immediately surrounding it are common to both the bronchial and intestinal tumors. Therefore the author designates these bronchial tumors as carcinoid of the bronchial mucous membrane, at least until their origin is more definitely established.—*Über gutartige Bronchial-tumoren (Cylindrome und Carcinoid), H. Hamperl, Virchow's Arch., August, 1937, ccc, 46.*—(*C. L. D.*)

Bronchial Carcinoma.—The disproportion between the number of cases of carcinoma of the bronchus diagnosed and the number successfully treated is considered and the fact pointed out that a late diagnosis is of little or no importance from the standpoint of the patient as the newer advances in surgery are then of no avail. The symptoms of which the patient complains are often those of an advanced carcinoma, but when early symptoms of cough with or without sputum, blood-streaking, pain and wheezing are present they should be carefully interpreted and the possibility of carcinoma of the bronchus kept in mind. Especial attention is called to the importance of wheeze due to partial obstruction of a bronchus as an early symptom. Every diagnostic means should be used to differentiate carcinoma from tuberculosis, pneumonitis, bronchiectasis, or pulmonary abscess, especially in the group in which there is no roentgenological sign of obstruction or tumor. Early X-ray studies should be obtained. Inspiratory and expiratory roentgenograms to demonstrate the presence or absence of obstructive emphysema are of value. Bronchoscopy should be freely employed when the lesion is centrally located as it is the most valuable diagnostic measure available, for not only can the area involved be seen, but a biopsy may be obtained and fixity and rigidity of the bronchial wall determined. A series of 143 cases of bronchial carcinoma diagnosed during life or at autopsy is reported; of these, 98 were definitely diagnosed by bronchoscopy and biopsy. The latter lesions originated in the larger bronchi and were easily seen. Three cases were diagnosed from tissue obtained following an exploratory thoracotomy and five were found when surgical drainage was done for what was thought to be pulmonary abscess. Rigidity and fixation of the bronchial wall was noted in 23 cases. In only 14 were the bronchoscopic findings inconclusive and diagnosis made at autopsy. In cases where the growth does not arise or intrude into the larger bronchi, repeated bronchoscopies will frequently aid in making the correct diagnosis. When bronchoscopy reveals nothing definite, lung mapping with lipiodol is of value. The roentgenologist should at all times be acquainted with the clinical findings and history, and close coöperation exist between clinician, roentgenologist and bronchoscopist so that more early diagnoses may be made. Dr. Rigler discussed this paper and called attention to the possibility of false positive diagnoses which might result in unnecessary drastic surgery.—*Carcinoma of the Bronchus, L. H. Clerf, Radiology, April, 1937, xxviii, 438.*—(*G. F. M.*)

Carcinoma in Anthracosilicosis.—A high incidence of bronchial and pulmonary carcinoma would be anticipated in a condition such as anthracosilicosis where chronic irritation from silica and dust is present. However, few cases of bronchogenic carcinoma associated with anthracosilicosis have been reported. In a two-year period, 174 cases of anthracosilicosis were treated. Four out of 36 cases in this group that came to autopsy were found to have primary bronchial carcinoma. The clinical and pathological findings of these four cases are briefly reviewed: The chief symptoms were dyspnoea, haemoptysis, cough, expectoration and pain in the chest. Except for haemoptysis the symptoms are those usually experienced in pure anthracosilicosis, but haemoptysis often occurs in the presence of an associated tuberculosis. Repeated negative sputum examinations aroused the suspicion that malignancy rather than tuberculosis was the complicating condition. In these cases the X-ray findings were those of atelectasis, but the anthracosilicosis made an X-ray diagnosis of tumor difficult. All showed extensive destruction of the bronchial cartilages resulting in the collapse of the involved bronchi. Extensive cavitation was present in three cases, but there were none of the symptoms associated with pulmonary abscess, probably due to the slow absorption of toxic material. The outstanding histopathological feature was the extensive infiltration and destruction of the anthracosilicotic nodules by tumor cells. The central avascular portions of the nodules were free of tumor cells, but the more cellular peripheries were thickly infiltrated by them. In spite of the marked fibrosis present, the perivascular and the peribronchial lymphatics revealed tremendous dilatation and engorgement by tumor cells. The spread of the carcinoma through alveolar pores and interlobar adhesions was also observed. Tumor thrombi were seen in many blood vessels. Cavities were doubtless the result of these pulmonary infarctions or poor bronchial drainage. Central necrosis was present in some of the tumor nodules. It is suggested that anthracosilicosis contributed indirectly in the development of these malignancies.—*Carcinoma of the Bronchus in Association with Anthracosilicosis, R. Charr, Amer. Jour. Med. Sc., October, 1937, cxciv, 535.*—(*G. F. M.*)

Pneumonoconiosis and Tuberculosis in Coal Miners.—From among the coal miners of South Wales, 100 cases of pneumonoconiosis were selected. There were 47 hard-ground (stone) workers, 43 hewers, and 3 without detailed history. The disease occurred more frequently among the hard-ground workers, 47 per cent, although they are a small minority of the total men employed in the industry. Bronchitis was a common complaint in the previous history of the men. There was a history of pleurisy in 29 per cent and of these 14 per cent were tuberculous, 2 had tuberculous effusions. A history of influenza was obtained in 22 per cent. A single attack precipitated dyspnoea in 20 per cent of the cases. A single attack had no significance but among those having repeated attacks, 22 per cent were tuberculous. Dyspnoea was the most frequent present symptom. Its severity did not always correspond with the degree of opacity in the X-ray films. Progress of the dyspnoea was rapid while the men worked, the average time from onset to disability was 2 years. Sudden increase in dyspnoea in a miner not working suggested tuberculosis. Clubbing of fingers was present in 15 per cent and the pulse rate was generally higher than normal. The majority of the men had a chest expansion of one-half to one inch. Meagre physical signs were present in the chest. On the X-ray films fine mottling was present in 26 cases, medium mottling in 53, and large and gross mottling in 21 cases. Eight cases showed definite signs of tuberculosis, all with positive sputum. Twenty-two suggested tuberculosis and 4 of these had positive sputum. Von Bonsdorff's modification of the Arneth index was lower than normal and sedimentation rate more rapid. Tubercle bacilli were found in the sputum in 12 per cent of the cases. Blood tests, X-ray findings and symptoms indicated tuberculosis in 23 per cent of cases in addition to the 12 per cent definite cases, although the evidence is not conclusive. The total of 35 per cent of tuberculosis is a high incidence. Of the 12 definite cases, 7 had no symptoms, suggesting that the disease was somehow modified. The presence of coal dust is suggested as the modifying factor and leads to the failure to recognize the tuberculosis or to mistake it for bronchitis. —*Pneumonoconiosis in South Wales Coal Miners and Its Relation to Tuberculosis, P. K. Sen, Jour. Indust. Hyg. & Toxicol., June, 1937, xix, 225.*—(*L. F. B.*)

Pulmonary Fibrosis in Miners.—Of various dusts inhaled in mines the stone dusts are undoubtedly most dangerous but there is no evident relation between the duration of exposure to stone dust and the intensity or rapidity of evolution of pulmonary lesions, nor are the effects uniform and constant. It is not unusual to find, in miners who have given up work in stone for a number of years, lesions of the pneumono-

coniosis type appear. The generic term "pulmonary fibrosis" seems preferable to the more restricted one of "silicosis." These fibrous lesions usually begin in the central part of the right lung and rapidly become bilateral and symmetrical. The tubercle bacillus is often discovered during the course of the disease and in certain negative cases is demonstrable by guinea-pig inoculation. Patients with advanced pulmonary fibrosis often die of pulmonary tuberculosis although sometimes cardiac decompensation supervenes. Necropsy may reveal coexistent pulmonary tuberculosis, impossible to diagnose before death; apparently purely fibrotic lesions, inoculation of tissues from which into a guinea pig eventually produces tuberculosis. Tuberculous contacts may be discovered either in the home or in the mine.—*Some Remarks on Pulmonary Fibrosis in Miners, Croizier and E. Martin, Tubercle, February, 1937, xviii, 206.*—(*A. P.*)

Coal Miners' Chest Diseases in Scotland.—Among 64 miners suffering from chest affections, ranging 20 to 67 years of age, 42 per cent were found to have pneumonoconiosis, 34 per cent chronic bronchitis, 17 per cent pulmonary tuberculosis, 5 per cent bronchial asthma and 2 per cent bronchiectasis. With one exception all had worked underground since leaving school, drilling, stripping and brushing being the main occupations. Fifty-nine per cent of the cases were totally incapacitated. Cyanosis was present in 22 per cent, clubbing of fingers or incurving of the nails in 28 per cent and a typical barrel-shape was noticed in 31 per cent. Chest expansion was often as low as half an inch. Valvular cardiac disease was absent but evidence of myocardial decompensation was present in 9 per cent of the cases. Dyspnoea was a prominent symptom and haemoptysis had occurred in 7.8 per cent of the cases. In 5 per cent of the cases the sputum was positive for tubercle bacilli. On X-ray examination, 12 per cent showed tuberculous changes, 46 per cent showed diffuse occupational fibrosis and 30 per cent showed peribronchial fibrosis. The family histories of this group compared with those of 64 healthy miners showed the incidence of pulmonary affection greater (42.2 per cent) among the blood relations of the affected miners than among the blood relations of the healthy miners (18.8 per cent). Miners have been known to have worked 20 or more years without showing clinical signs of respiratory disease. Chest X-rays of two such miners showed marked pneumonoconiosis, yet the men were not incapacitated. These observations suggest that the X-ray appearances of pneumonoconiosis are not in themselves conclusive evidence that this condition is a disease entity. In order to lower the incidence of respiratory diseases the workers should be recruited from families in whom bronchitis and other chest complaints are absent.—*A Study of Chronic Chest Diseases among Coal Miners in Shotts, Scotland, J. A. M. Hall, Jour. Indust. Hyg. & Toxicol., November, 1937, xix, 506.*—(*L. F. B.*)

Key to Abstractors

A. A. E.: Adrian A. Ehler, Ann Arbor, Michigan.
A. E. M.: Aaron E. Margulis, Mt. Morris, New York.
A. P.: Andrew Peters, Springfield, Massachusetts.
C. L. D.: Charles L. Dunham, Chicago, Illinois.
E. M. J.: Edna M. Jones, Northville, Michigan.
G. F. M.: Gertrude F. Mitchell, Detroit, Michigan.
L. F. B.: Lauren F. Busby, Northville, Michigan.